

L Number	Hits	Search Text	DB	Time stamp
9	119	flavivirus and vero	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/02/19 15:05
10	13555	(flavivirus and vero) and yellow fever	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/02/19 15:05
11	119	(flavivirus and vero) and (yellow fever virus)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/02/19 15:06
12	92	((flavivirus and vero) and (yellow fever virus)) and vaccine	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/02/19 15:19
13	2	((((flavivirus and vero) and (yellow fever virus)) and vaccine) and yf17d	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/02/19 15:19

TI Preparation of inactivated viral vaccines  
IN Wieseahn, Gary P., Alameda, CA, United States  
Creagan, Richard P., Alta Loma, CA, United States  
Stevens, David R., Fremont, CA, United States  
Giles, Richard, Alameda, CA, United States  
AB Vaccines employing inactivated viruses having improved retention of antigenic characteristics are prepared by psoralen-inactivation of the live virus in a non-oxidizing atmosphere. By excluding oxygen and other oxidizing species from the inactivation medium, degradation of the antigen characteristics resulting from irradiation with ultraviolet light is largely prevented. The resulting inactivated viruses are employed in vaccine preparations for the inoculation of susceptible hosts to inhibit viral infection.  
AN 87:65285 USPATFULL  
TI Preparation of inactivated viral vaccines  
IN Wieseahn, Gary P., Alameda, CA, United States  
Creagan, Richard P., Alta Loma, CA, United States  
Stevens, David R., Fremont, CA, United States  
Giles, Richard, Alameda, CA, United States  
PA Advanced Genetics Research Institute, Oakland, CA, United States (U.S. corporation)  
PI US 4693981 19870915 <--  
AI US 1985-785354 19851007 (6)  
DCD 20021008  
RLI Continuation-in-part of Ser. No. US 1983-563939, filed on 20 Dec 1983, now patented, Pat. No. US 4545987 And a continuation-in-part of Ser. No. US 1984-592661, filed on 23 Mar 1984, now abandoned  
DT Utility  
EXNAM Primary Examiner: Rose, Shep K.  
LREP Rowland, Bertram I.  
CLMN Number of Claims: 9  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1219  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 38 OF 81 USPATFULL  
TI Flavivirus recombinant poxvirus vaccine  
IN Paoletti, Enzo, Delmar, NY, United States  
Pincus, Steven E., East Greenbush, NY, United States  
AB What is described is a recombinant poxvirus, such as vaccinia virus,  
fowlpox virus and canarypox virus, containing foreign DNA from  
flavivirus, such as Japanese encephalitis virus, **yellow**  
**fever virus** and Dengue virus. In a preferred  
embodiment, the recombinant poxvirus generates an extracellular  
particle  
containing flavivirus E and M proteins capable of inducing neutralizing  
antibodies, hemagglutination-inhibiting antibodies and protective  
immunity against flavivirus infection. What is also described is a  
vaccine containing the recombinant poxvirus for inducing an  
immunological response in a host animal inoculated with the vaccine.  
AN 96:38606 USPATFULL  
TI Flavivirus recombinant poxvirus vaccine  
IN Paoletti, Enzo, Delmar, NY, United States  
Pincus, Steven E., East Greenbush, NY, United States  
PA Virogenetics Corporation, Troy, NY, United States (U.S. corporation)  
PI US 5514375 19960507 <--  
AI US 1991-714687 19910613 (7)  
RLI Continuation-in-part of Ser. No. US 1991-711429, filed on 6 Jun 1991,  
now abandoned And a continuation-in-part of Ser. No. US 1991-713967,  
filed on 11 Jun 1991, now abandoned which is a continuation-in-part of  
Ser. No. US 1991-666056, filed on 7 Mar 1991, now abandoned , said Ser.  
No. US -711429 which is a continuation of Ser. No. US 1990-567960,  
filed on 15 Aug 1990, now abandoned  
DT Utility  
EXNAM Primary Examiner: Sidberry, Hazel F.; Assistant Examiner: Tuscan,  
Michael  
LREP Curtis, Morris & Safford  
CLMN Number of Claims: 22  
ECL Exemplary Claim: 1  
DRWN 19 Drawing Figure(s); 16 Drawing Page(s)  
LN.CNT 2530  
CAS INDEXIN

L6 ANSWER 8 OF 17 MEDLINE  
 TI Attenuation of wild-type yellow fever virus by passage in HeLa cells.  
 AU Barrett A D; Monath T P; Cropp C B; Adkins J A; Ledger T N; Gould E A; Schlesinger J J; Kinney R M; Trent D W  
 AB During the 1960s three different research groups reported that passage of wild-type yellow fever (YF) virus [strain Asibi (YF-Asibi)] in HeLa cells resulted in attenuation of the virus for monkeys so that the virus no longer caused viscerotropic disease. We have repeated and extended this observation to analyse the process of attenuation of YF virus during cell culture passage. A large plaque (LP) variant of YF-Asibi virus became attenuated for both monkeys and mice following six serial subcultures in HeLa cells (YF-Asibi-LP HeLa p6). Thus, attenuation was probably due to a genetic change in the virus population rather than to selective enrichment of a pre-existing variant of YF-Asibi-LP virus. No evidence was obtained to implicate defective interfering particles in the attenuation process. Comparison of the YF-Asibi-LP viruses before and after passage in HeLa cells, using a panel of envelope protein-reactive monoclonal antibodies (MAbs), showed that MAbs which specifically neutralize YF-Asibi-LP virus, and not YF 17D-204 vaccine virus, also neutralized YF-Asibi-LP HeLa p6. This indicated that the epitopes involved in the biological process of neutralization were not altered during attenuation. However, two MAbs that recognize envelope protein epitopes did distinguish between HeLa- and non-HeLa-passaged YF-Asibi-LP virus. One of these (MAb 117) which is YF wild-type-specific, recognized YF-Asibi-LP virus but not YF-Asibi-LP HeLa p6 virus, whereas the other (MAb411), which is YF vaccine-specific, recognized YF-Asibi-LP HeLa p6 virus but not YF-Asibi-LP virus. These results suggest that antigenic changes in the viral envelope protein may determine the relative virulence or attenuation of YF virus.

AN 91037961 MEDLINE  
 DN 91037961 PubMed ID: 2230735  
 TI Attenuation of wild-type yellow fever virus by passage in HeLa cells.  
 AU Barrett A D; Monath T P; Cropp C B; Adkins J A; Ledger T N; Gould E A; Schlesinger J J; Kinney R M; Trent D W  
 CS Department of Microbiology, University of Surrey, Guildford, U.K.  
 SO JOURNAL OF GENERAL VIROLOGY, (1990 Oct) 71 ( Pt 10) 2301-6.  
 Journal code: I9B; 0077340. ISSN: 0022-1317.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199012  
 ED Entered STN: 19910208  
 Last Updated on STN: 19970203  
 Entered Medline: 19901204

QR1. J6

L6 ANSWER 11 OF 17 MEDLINE  
 TI Conditions for haemolysis by flaviviruses and characterization of the haemolysin.  
 AU Cammack N; Gould E A  
 AB The 17D vaccine strain of yellow fever virus (**YF 17D**) was used to establish the optimal conditions for lysis of chick erythrocytes. Tissue culture-grown, polyethylene glycol-concentrated virus showed peak activity at pH 5.4 in citrate buffer when incubated at 37 degrees C. A further two- to fourfold increase in titre was obtained by pretreatment of the chick erythrocytes with 250 micrograms/ml trypsin. These conditions were also shown to be optimal for Japanese encephalitis (JE), West Nile (WN) and dengue-2 (den2) viruses. The ratio of haemagglutination (HA) titre to haemolysis (HL) titre approximated to unity, suggesting that the two functions are associated with the same molecule although as separable entities since selective inactivation of the HL activity of the virus was accomplished using 60 micrograms/ml trypsin. HL could be demonstrated at neutral pH if the chick erythrocytes were first subjected to treatment with acidic pH buffer. The effect on the virus envelope is thus not the sole contribution of a low pH environment to optimal HL. Hyperimmune rabbit antiserum prepared against purified **YF 17D** virions inhibited HA and HL if added before agglutination had occurred by the virus but when added after agglutination had taken place it showed specific anti-HL activity. Monoclonal antibodies that inhibited HA (HAI) by **YF 17D** did not inhibit HL (HLI) activity when applied after agglutination had taken place. Moreover, monoclonal antibodies specific for the 54K glycoprotein of YF virus but without HAI activity also had no effect on HL when added either before or after agglutination. As yet, we have been unable to identify a monoclonal antibody displaying specific anti-HL activity but all those directed against the 54K envelope glycoprotein possessing HAI activity showed HA to be a prerequisite for HL.  
 AN 86010270 MEDLINE  
 DN 86010270 PubMed ID: 2995565  
 TI Conditions for haemolysis by flaviviruses and characterization of the haemolysin.  
 AU Cammack N; Gould E A  
 SO JOURNAL OF GENERAL VIROLOGY, (1985 Oct) 66 ( Pt 10) 2291-6.  
 Journal code: I9B; 0077340. ISSN: 0022-1317.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198511  
 ED Entered STN: 19900321  
 Last Updated on STN: 19900321  
 Entered Medline: 19851108

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L7 ANSWER 9 OF 81 MEDLINE  
 TI Virulence and pathogenesis of **yellow fever**  
**virus** serially passaged in **cell culture**.  
 AU Converse J L; Kovatch R M; Pulliam J D; Nagle S C Jr; Snyder E M  
 AN 71267151 MEDLINE  
 DN 71267151 PubMed ID: 4998347  
 TI Virulence and pathogenesis of **yellow fever**  
**virus** serially passaged in **cell culture**.  
 AU Converse J L; Kovatch R M; Pulliam J D; Nagle S C Jr; Snyder E M  
 SO APPLIED MICROBIOLOGY, (1971 Jun) 21 (6) 1053-7.  
 Journal code: 6K0; 7605802. ISSN: 0003-6919.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 197110  
 ED Entered STN: 19900101  
 Last Updated on STN: 19970203  
 Entered Medline: 19711015

L7 ANSWER 6 OF 7 MEDLINE  
TI THE **GROWTH** OF ASIBI STRAIN **YELLOW FEVER**  
VIRUS IN TISSUE CULTURES. II. MODIFICATION OF VIRUS AND CELLS.  
AU HARDY F M  
AN 64001668 MEDLINE  
DN 64001668  
TI THE **GROWTH** OF ASIBI STRAIN **YELLOW FEVER**  
VIRUS IN TISSUE CULTURES. II. MODIFICATION OF VIRUS AND CELLS.  
AU HARDY F M  
SO JOURNAL OF INFECTIOUS DISEASES, (1963 JUL-AUG) 113 9-14.  
Journal code: IH3. ISSN: 0022-1899.  
CY United States  
DT Journal  
LA English  
FS OLDMEDLINE  
EM 196401  
ED Entered STN: 19990716  
Last U

L7 ANSWER 5 OF 7 MEDLINE  
TI **GROWTH** OF 17D **YELLOW FEVER** VIRUS AND FACTORS  
INFLUENCING ITS TRANSMISSION WITHIN CELL CULTURES IN VITRO.  
AU LITWIN J  
AN 64143518 MEDLINE  
DN 64143518  
TI **GROWTH** OF 17D **YELLOW FEVER** VIRUS AND FACTORS  
INFLUENCING ITS TRANSMISSION WITHIN CELL CULTURES IN VITRO.  
AU LITWIN J  
SO ACTA PATHOLOGICA ET MICROBIOLOGICA SCANDINAVICA, (1964) 61 605-18.  
Journal code: 100. ISSN: 0365-5555.  
CY Denmark  
DT Journal  
LA English  
FS OLDMEDLINE  
EM 196412  
ED Entere



L7 ANSWER 2 OF 7 MEDLINE DUPLICATE 2

TI **Growth** of 17D **yellow fever** virus in a  
macrophage-like cell line, U937: role of Fc and viral receptors in  
antibody-mediated infection.

AU Schlesinger J J; Brandriss M W

AB Growth characteristics of 17D yellow fever virus (17D-YF) and conditions  
for infection were studied in U937, a macrophage-like, Fc  
receptor-bearing  
continuous human cell line. Antibody to 17D-YF was obtained by  
immunization of normal subjects with 17D-YF vaccine. Cells were infected  
in the presence or absence of immune whole sera or immunoglobulin  
fractions. Infection of U937 was temperature dependent; the yield of  
virus  
was variable but at low temperature viral titers were consistently higher  
when infection was established in the presence of antibody. Results of  
infectious center assays indicated that the increased yield of virus was  
largely or entirely due to an increase in the number of cells producing  
virus early in the course of infection. Enhancement of viral growth was  
mediated by IgG but not IgM fractions of immune sera. Trypsinization of  
U937 resulted in a 90 to 95% reduction of infection in the absence of  
antibody but in the presence of antibody viral titers were higher in  
trypsinized than in nontrypsinized cells. Antibody to 17D-YF, contained  
in  
the whole IgG fraction of sera, bound to U937 to mediate infection  
without  
first being complexed to virus. Preincubation of U937 with IgG1 but not  
IgG2 myeloma proteins abrogated antibody-mediated infection. This result  
is compatible with the known affinities of U937 Fc receptors for specific  
subclasses of IgG and provides evidence for the role of the Fc receptor  
in  
antibody-mediated enhancement of viral growth. Persistent infection  
characterized by a lack of detectable cytopathogenic effect was  
established in long-term cultures of U937. This pattern of infection  
might  
be related to the unique effectiveness of the 17D-YF vaccine.

AN 81240805 MEDLINE

DN 81240805 PubMed ID: 7252155

TI **Growth** of 17D **yellow fever** virus in a  
macrophage-like cell line, U937: role of Fc and viral receptors in  
antibody-mediated infection.

AU Schlesinger J J; Brandriss M W

SO JOURNAL OF IMMUNOLOGY, (1981 Aug) 127 (2) 659-65.  
Journal code: IFB; 2985117R. ISSN: 0022-1767.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Abridged Index Medicus Journals; Priority Journals

EM 198109

ED Entered STN: 19900316  
Last Updated on STN: 19900316  
Entered Medline: 19810915

QR 180.J6